DIASTOLIC AORTIC MURMURS WITHOUT VALVULAR LESIONS.

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Reprinted from the Edinburgh Medical Journal. *Edinburgh and London:* Young J. Pentland, *December*, 1903.

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Diastolic murmurs in the absence of valvular lesions present in their occurrence a notable contrast as regards their seat. Of comparative frequency in relation to the pulmonary orifice, they occur much less commonly at the aortic orifice. The reason for this is not far to seek. The aortic orifice is surrounded by such a resistant ring that its dilatation can only occur in consequence of degeneration changes and great pressure, while the origin of the pulmonary artery, although not nearly so often the seat of degenerative lesions, may be distended by a comparatively moderate force. This point was thoroughly investigated by me many years ago, and the results, which will be referred to in the sequel, are of extreme interest in view of the discordance between the incidence of regurgitation without valvular disease at the two great arterial orifices.

Aortie regurgitation without valvular lesion has, nevertheless, been from time to time recorded since the days of Corrigan.[1] Aran has often been referred to as the author of similar observations, but although undoubtedly in his case it was a simple stretching of the aortic ring that caused the regurgitation, the valves were not perfectly healthy.[2] Chevers,[3] Bellingham,[4] Peacock,[5] Perls,[6] Besnier,[7] Coekle,[8] Pel,[9] Chauffard,[10] Finlayson,[11] Bouveret,[12] Renvers,[13] Klein,[14] Dombrowski,[15] Bonnet,[16] Pitt,[17] and Gairdner,[18] have all narrated instances of undoubted diastolic murmurs, apparently generated at the aortic orifice without valvular lesions. Quite recently Cabot and Locke have written an interesting paper [19] on the occurrence of diastolic murmurs without lesions of the aortic or pulmonary valves, in which, in addition to giving a short sketch of the literature of the subject, they furnish a description of four cases. To my mind, the facts of these four cases are not convincing of aortic diastolic murmurs. It must be admitted, and indeed it has been stated by me,[20] that an aortic diastolic murmur frequently has its maximum intensity to the left of the sternum; in instances of this kind the diagnosis of aortic, as opposed to pulmonary, regurgitation must depend upon the attendant symptoms. Yet in only one of the cases mentioned by Cabot and Locke could it be said that there was even possibly an aortic diastolic murmur.

My experimental observations led me to expect functional pulmonary incompetence in high pressure,[21] but it was many years before a case of the kind came under my observation. Many such instances have crossed my path since, and some of them, with full abstracts of the literature of the subject, have been recorded.[22] As Cabot and Locke state, no mention of diastolic

[1] *Edin. Med. and Surg. Journ.*, 1832, vol. xxxvii. p. 225.
[2] *Arch. de méd.*, Paris, 1842, tome xv. p. 265.
[3] *Guy's Hosp. Rep.*, London, 1842, vol. vii. p. 387.
[4] "Diseases of the Heart," Dublin, 1853, p. 152.
[5] "On Some of the Causes and Effects of Valvular Heart Disease," London, 1865, p. 48.
[6] *Deutsches Arch. f. klin. Med.*, Leipzig, 1869, S. 381.
[7] *Bull. Soc. anat. de Paris*, 1873, p. 233.
[8] "Contributions to Cardiac Pathology," London, 1881, p. 12.
[9] *Berl. klin. Wchnschr.*, 1881, S. 135.
[10] *Bull. Soc. anat. de Paris*, 1882, p. 188.
[11] *Brit. Med. Journ.*, London, 1885, vol. i. p. 426; and *Glasgow Med. Journ.* 1896, vol. xlv. p. 299.
[12] *Lyon méd.*, 1888, tome lviii. p. 153.
[13] *Charité-Ann.*, Berlin, 1888, Bd. xiii. S. 225.
[14] *Wien. med. Wchnschr.*, 1889, S. 703.
[15] *Rev. de méd.*, Paris, 1893, p. 757.
[16] *Lyon méd.*, 1897, tome lxxxv. p. 505.
[17] *Trans. Path. Soc.*, London, 1898, vol. xlix. p. 46.
[18] *Glasgow Hosp. Rep.*, 1900, vol. ii. p. 1.
[19] *Johns Hopkins Hosp. Bull.*, Baltimore, 1903, vol. xiv. p. 115.
[20] "Diseases of the Heart and Aorta," Edinburgh and London, 1898, p. 496.
[21] *Edin. Med. Journ.*, 1880, vol. xxv. p. 979.
[22] *Edin. Hosp. Rep.*, 1894, vol. ii. p. 320; and "Diseases of the Heart and Aorta," 1898, p. 580.

murmurs without aortic dilatation or valvular lesion is made in my work on the heart—in fact, it must be confessed that my position was one of suspended judgment until recent years; hence the remark: "The aortic orifice is supported by an extremely resistant fibrous ring which seems almost incapable of dilatation under ordinary circumstances, so that regurgitation from the aorta into the left ventricle can only take place by means of absolute incompetence of the cusps, not through any increase in the size of the orifice."[1] More extended observation, however, has led me to adopt an opinion which coincides, except as regards the question of frequency, with the view recently expressed by Babcock: "There is a form of aortic insufficiency which, although not due to valvular defect, yet presents the same clinical features as the organic form, and is so frequently encountered that it may here be briefly dwelt upon. This is a relative incompetence of the semilunar valve, and its causes are found in conditions that predispose to stretching of the ventricular wall and of the basal ring of the aorta."[2] Two recent examples of this type of aortic regurgitation are of sufficient interest to merit narration.

Case 1.—A. B., æt. 30, a bank clerk, was recommended by Dr. Murray to Ward 29 of the Royal Infirmary, 6th May 1903, complaining of "an itching skin disease."

His family history was bad, his father dying of chronic alcoholism; his mother had a tendency to gout; her father and one uncle and three aunts had died of apoplexy; the patient also had an aunt and two cousins confined in different asylums, and there was a history of tumour in the father's family. No account of tubercle or specific infection could be obtained, but his only sister was away in Canada on account of her lungs, which were weak.

The patient's surroundings at work were excellent, and his hours were not long; his home was very comfortable, and his diet good; he was almost a teetotaller, and was not a heavy smoker. He had been an enthusiastic athlete, but had given up athletics in great part during the last three years, as he did not feel strong, though still in good health; he was in the habit of sleeping between 6 and 9 every evening, as he felt tired after the office work.

His previous health had been good, except that, up till two years of age, he suffered from bronchitis, and as a schoolboy had extremely bad headaches, which necessitated his stopping work for days together; these had completely left him for many years. There was no history of specific infection, or of any fever.

Two years before the present illness, he had been refused for insurance, as he was told he had chronic Bright's disease, though he was feeling perfectly well at the time, and continued so to within six weeks of admission, when a slight red papular and extremely itching rash broke

[1] "Diseases of the Heart and Aorta," 1898, p. 487.
[2] "Diseases of the Heart and Arterial System," New York and London, 1903, p. 281.

out on the inner side of the thighs; this spread all over the body in the course of the next three days; the rash was extremely irritating, and Dr. Murray of Leith, who was called in, sent the patient to me at the Royal Infirmary.

When admitted, the patient was a tall, well-made, heavily built man; his face appeared slightly puffy, and he was somewhat anæmic. The skin was nearly covered with an eruption, consisting of very small red papules which, on the legs and thighs and elbows, had coalesced; the surface was shrouded by desquamating epidermis, under which the cutis was a bright red; the itching was intense, and had prevented his sleeping some days before admission. There was no œdema to be made out anywhere.

As regards the *urinary system*, the patient had noticed no polyuria or increased frequency of micturition. On the day after admission, the quantity passed was 78 oz.; the colour was very pale straw, somewhat opalescent; the reaction was acid; the sp. gr. was 1008; the urea, 3·6 grs. to the ounce; the urine also contained 2·18 grs. of albumin to the ounce. There was a slight deposit which showed epithelial cells, and a few leucocytes, and also a few leucocyte and hyaline tube casts.

The *respiratory system* showed nothing abnormal, both lungs being clear all over, and the breath sounds vesicular over both lungs, with expiration slightly prolonged. There were no signs of *alimentary* disturbance, excepting that the tongue was slightly furred and the appetite poor, and the thirst somewhat excessive.

There were no subjective symptoms in connection with the *circulatory system*. The præcordia were somewhat bulging, and the apex beat was seen in the sixth interspace, 5 in. from the mid-line; there was some indrawing of the surrounding interspaces at each systolic impulse. The epigastrium was pulsating, and the veins of the neck were very turgid, distended, and pulsating forcibly, though there was no systolic collapse noted. The apex beat was diffuse, but forcible on palpation, and a slight systolic thrill could be felt at that spot. The right border of the heart was $2\frac{1}{2}$ in. from mid-line in the third space, and the left was 3 in. from mid-line in the third, and $5\frac{3}{4}$ in. in the sixth space. On auscultation in the aortic area, a hard harsh systolic murmur was heard, which was propagated into the carotids. The second sound was impure, and was followed by a soft blowing diastolic murmur which was best heard over the sternum, and was hardly propagated at all. Both sounds in the other areas were loud and closed.

The arterial walls were somewhat thickened; the pulse rate was 90 per minute and quite regular in time, except that it missed a beat about once in every 50. The volume of the wave was very variable, as it fell away considerably during inspiration, and became greater on expiration; the pressure was also raised during expiration, and fell on inspiration; in short, it was a typical example of the paradoxical pulse.

The diagnosis arrived at was that of chronic nephritis, with aortic obstruction and regurgitation, as well as probable mediastino-pericarditis, and the skin condition was classed as an exfoliative dermatitis.

The patient improved, as far as the disease for which he came under treatment was concerned, until 8th May, when he complained of headache; in the morning, and later in the day, the breathing became stertorous, and uræmia was evidently setting in. This proved fatal on 13th May, in spite of diaphoresis, catharsis, and intravenous saline injections. About 20 oz. of blood were removed from a vein in the forearm, and when the vein was opened the blood spurted out to a distance of quite 2 ft.; the stream was much more forcible on expiration than on inspiration; the pulse pressure was high, and the rate regular up till just before death.

A post-mortem examination was made on 14th May by Dr. Beattie.

Rigor mortis was general, the body well nourished, and post-mortem lividity was well marked, especially posteriorly. On opening the chest, both the pleural cavities were found free of adhesions. The heart weighed 2 lb. 1 oz., the pericardium was adherent all over the surface. The heart as a whole was much hypertrophied, the left ventricle measuring 1 in. in thickness at its thickest part, and the right ventricle $\frac{3}{8}$ in. All the valves were found competent, the aortic segments were thickened at their free margins, and the aorta showed patches of atheroma. Round the base of the great vessels a calcareous band about $\frac{1}{2}$ in. thick was found, which surrounded the aorta and pulmonary artery, and compressed them. The lungs showed marked chronic venous congestion, and œdema of the bases. No signs of tubercle were found in them. The anterior margins of all the lobes were emphysematous. The abdominal viscera showed marked venous congestion; the liver weighed 4 lb. 15 oz., and the spleen 8 oz. The left kidney weighed 9 oz., the capsule was thickened and adherent, and the surface granular and mottled; the organ showed an extreme degree of interstitial nephritis with glomerular changes. The vessels were very much thickened. The right kidney weighed 7 oz., and showed the same changes in a slightly less degree.

In this case the dense band surrounding the aorta and pulmonary artery caused considerable displacement, as well as constriction, and it seems to my mind quite clear that the diastolic murmur was produced, not by stretching of the orifice (which, indeed, was negatived by the result of the post-mortem examination), but by faulty adaptation of the aortic cusps. The systolic murmur was no doubt largely due to some dilatation of the vessel beyond the constriction.

Case 2.—C. D., æt. 37, baker, was recommended to Ward 29, 12th March 1901, on account of a swelling in front of the chest.

The family history was fairly good. His father was alive and well. His mother had died about ten years before of bronchitis. He had two brothers and four sisters alive and well, and he had also had two sisters who died in childhood. His wife had been confined twice; the first

child was alive and well, but the second confinement had been a miscarriage. The patient's appetite was good, and he took all manner of food easily and without discomfort. For over fifteen years he had been a habitual drunkard, almost nightly taking beer or whisky, or both, to excess. He was not a smoker, but took large quantities of snuff. His home was comfortable, and he had always had enough food. In occupation he was a baker, and when working at his trade he was subjected occasionally to considerable heat, but the work was not very heavy. He was occasionally out of work, and for the last three years had been almost constantly so; during these periods he worked upon "the steamers" as a stevedore, where his work was very heavy, involving the lifting of great weights.

At about the age of 17, the patient had had an attack of typhoid fever. About twenty years before he entered the infirmary he had contracted syphilis; and he had twice had gonorrhœa. When he was 22 years of age, he had a severe fall upon his chest, but felt no subsequent ill effects. He was once severely scalded over the back and shoulder by boiling starch. For five or six years he had suffered from a weak chest, and had also had bronchitis and been treated for this. About a year before admission, the patient, standing behind a man who was stopping an engine, was violently struck upon the chest by the man's elbow as it was driven back by the brake. He suffered great pain on that and the following day, but did not stop work. Three days later he felt a "pumping" over his chest, but no pain; and not long afterwards he noticed a swelling. He felt no inconvenience from it, except that, if undergoing an exceptional strain, he experienced a sharp pain in his chest, which compelled him to stop work for a short time. He did not consult a doctor, and it was only incidentally that Dr. R. J. Johnston saw it, and sent him in to the ward.

The patient was apparently strong and well nourished. There was no appearance of emaciation, anæmia, cyanosis, jaundice, or dropsy, and there was no clubbing of the fingers or curving of the nails. On his face were some spots of acne. On both legs were some old scars, which he said were the result of boils. He had no special attitude. His height was 5 ft. $4\frac{1}{2}$ in. The temperature varied from 97° to 98°.

Circulatory system.—The patient experienced no pain, unless on severe exertion, or unless the projection were pressed. He had no palpitation or faintness, but on severe exertion the pain in his chest was accompanied by some breathlessness. On inspection, a tumour was seen about the size of a pigeon's egg, opposite the junctions of the third and fourth costal cartilage with the sternum, and slightly to the left of the midsternal line. The skin was strong and firm over the swelling. Below this swelling was a slight depression. The apex beat was not visible. The whole præcordia visibly heaved or throbbed, and the swelling pulsated regularly. On palpation, a heaving or pulsation of the swelling was very marked. This was expansile in character, and had a perceptibly double beat, giving a thrill-sensation to the hand. The heaving of the præcordia might also be felt. The apex beat was perceptible in the fifth interspace just internal to the mammary line, but was not very clearly defined. On percussion, superficial cardiac dulness began at the lower border of the third rib, and extended downwards to

the sixth. The right border of the heart was 2 in. to the right of the midsternal line. The left border was $4\frac{1}{2}$ in. from that line. On auscultation in the aortic area, the first sound was replaced by a harsh systolic murmur; the second sound was clear and slightly accentuated: in the mitral area, the first sound was accompanied by a harsh systolic movement; the second sound was closed: in the pulmonary area, a systolic murmur accompanied the first sound; the second sound was clear: in the tricuspid area, the first sound was concealed by a loud systolic murmur. Over the greater part of the præcordia the systolic murmur might be heard, and most distinctly over the swelling. The pulse varied from 72 to 80, was regular in rhythm, and of moderate pressure. Both radial arteries showed equal pulsation. A capillary pulsation could be made out on the forehead.

As regards the *alimentary system*, the lips and gums showed no abnormal appearances. The teeth were irregular, but fairly good. The tongue was slightly tremulous, but otherwise healthy. The mouth was moist, and the fauces showed nothing abnormal. His appetite for food and drink was good. He had no pain on swallowing. After a full meal he often had a feeling of oppression, and he suffered much from flatulence. His bowels were regular. He had occasionally suffered from vomiting, but only after a heavy drinking bout. On inspection, the abdomen was seen to be considerably distended, but on palpation did not feel very tense. Neither liver nor spleen could be felt, the parietes being apparently thickened. There was no tenderness on palpation. On percussion, the upper border of the stomach was found to be in the parasternal line, at the fifth rib; the lower border about midway between umbilicus and xiphisternum. The liver reached, in the mammary line, from the fourth rib above to $1\frac{1}{4}$ in. below the costal margin.

With reference to the *hæmopoietic system*, there were no enlarged lymphatic glands or vessels present.

Respiratory system.—The frequency of the breathing was 22. The rhythm was regular and the type abdomino-thoracic. The patient breathed naturally, and had no sensation of discomfort. He had a slight cough with a little thin watery sputum, but it troubled him very slightly. He said that four years ago his voice left him for a short time, and that it had done so occasionally since. His voice was somewhat husky, but he could speak loudly and clearly if desired. On inspection, he had a well-formed deep chest, with a small depression at the lower end of the sternum. Both sides of his chest expanded equally. On palpation, the vocal fremitus was found to be more distinct on the left side than on the right. On percussion, the apex of the right lung was $1\frac{1}{2}$ in., of the left 1 in., above the clavicle. The lower border was at the sixth rib in the mammary line. Posteriorly it was, in the scapular line, at the tenth rib. On auscultation, the breath was of harsh vesicular type. The vocal resonance was more distinct on the right than on the left. There were no abnormal sounds audible.

Integumentary system.—There was no pain, itching, or other uneasiness in the skin, which was moist but not wet. There was no emaciation, obesity, or œdema. There was no emphysema; on the face were a few spots of acne.

Urinary system.—No pain or uneasiness was felt in the act of micturition, which was of ordinary frequency. Urine in quantity, 45 oz.; colour, light, and with a greenish tinge, very faint; specific gravity, 1031; reaction, alkaline. No albumin, blood, sugar, or deposits.

As regards the *nervous system*, there was no peripheral irritability, and no alteration or abnormality in intelligence, temper, or reasoning. He could distinguish pain, heat, and cold; his sensibility to touch was good. The muscular sense was unimpaired. The sight was good, the pupils reacted to light, and neither was dilated. His hearing, taste, and smell were quite good. He swallowed easily. His micturition and defæcation showed nothing abnormal. Both plantar reflexes were present and somewhat exaggerated. No abdominal or epigastric reflexes. The cilio-spinal reflexes were present and equal. The knee-jerks were present and exaggerated. The triceps response was more easily elicited in the left arm than the right. The supinator responses could not be elicited. No clonus could be obtained in wrists, ankles, or patellæ. The voluntary movements were quite good. He co-ordinated perfectly. The electric irritability was normal. With reference to the vasomotor and nutritive functions, there were no local congestions, pallor, œdema, inflammation, sloughing, or wasting, and there was no unusual perspiration. The intelligence was remarkably good. There were no illusions, hallucinations, delusions, torpor, or coma. His attention and memory were good. His speech and sleep were unimpaired. There was no pain, swelling, or effusion in any joint, and he moved them easily.

The diagnosis was that of an aneurysm arising from the origin of the ascending aorta.

The patient improved considerably under treatment, and was allowed to leave the infirmary, 16th September. He returned, however, on the 16th of October, and again remained under treatment until 10th February, when he was for the second time allowed to leave, after once more undergoing considerable improvement. He was again in hospital from the 19th of February till the 22nd of March 1902, and from 4th July till 11th October 1902. During this latter period there was observed for the first time, a diastolic murmur over the upper part of the sternum and around the pulsating projection. The occurrence of this murmur led to considerable discussion amongst the members of my staff, and we were at a loss to decide whether it was due to implication of the cusps themselves, to a stretching of the aortic ring, or to a faulty adaptation of the cusps. On the whole, the latter appeared to be the most probable explanation. For the last time, the patient entered the ward on 1st July 1903, and remained under observation until his death on 20th August. During this time, although most diligently sought for, the diastolic murmur was not present, and it therefore seemed probable that the explanation at which we arrived, *i.e.* that of simple faulty apposition of the aortic cusps on account of displacement, was correct.

The post-mortem examination was conducted on 21st August

by Dr. Stuart M'Donald, from whose notes the following abstract has been obtained:—

Post-Mortem Report: C. D., æt. 39, Ward 29; died 20th August; *sectio* 21st August 1903.

External appearances.—Rigor mortis general; general pallor of body. Over the upper part of the sternum there is a perforation of skin and bone, the bone being destroyed to the extent of 2 in. in the transverse direction by $1\frac{3}{4}$ in. in the vertical. The opening lies between the levels of the first and lower border of the third costal cartilage; some recent blood clot lies in the opening.

Thorax.—On opening the thorax, a large saccular aneurysm is found, on the ascending and part of the transverse aortic arch; the aneurysm has perforated anteriorly as above. The anterior wall of the sac is densely adherent to the posterior aspect of the upper part of the sternum. On opening the sac, the aneurysm is found to measure about $3\frac{3}{4}$ in. from above downwards, and about $4\frac{3}{4}$ in. from side to side. The aneurysm extends from just above the aortic sinuses to just beyond the opening of the left subclavian. The left posterior aortic sinus is distinctly dilated, but does not form part of the wall of the aneurysm itself. The cavity of the aneurysm is divided into two main compartments by a projecting ridge or spur, best seen on the anterior wall, where it projects fully $\frac{3}{4}$ in. inwards. This ledge springs from the wall about 1 in. above the aortic cusps. There are numerous small secondary sacculations in the cavity. The lining of the sac shows patches of atheromatous degeneration with calcareous change throughout. The aorta at its commencement and beyond the aneurysm shows extensive atheroma. The anterior of the two main compartments is much thinner walled than the posterior, the aneurysm bulging mainly forward and to the right. The wall gradually thins and disappears at the edges of the perforation in the bone. The other main compartment bulges inwards towards the concavity of the arch. The opening of the sac anteriorly is blocked by recent red thrombus. Here and there on the wall elsewhere are some small masses of ante-mortem and mixed thrombus, best seen in the secondary sacculations of the anterior compartment. There is, however, on the whole, very little true ante-mortem thrombosis. The aneurysm, extending as it does mainly forwards, upwards, and to the right, does not appear to be causing special pressure on any important structure. The root of the right lung does not appear to be pressed on.

On opening the pericardium, about 3 oz. of clear fluid is found in the sac. There is no pericarditis.

The heart is left attached to the specimen and not weighed. The aortic valve appears to be very slightly incompetent; the cusps show some chronic thickening. The mitral, tricuspid, and pulmonary cusps show no special change. The left ventricle is not hypertrophied. The myocardium shows extensive diffuse fatty degeneration.

The lungs are pale. There is some emphysema of both upper lobes and outer margins of both lungs. On section there is slight congestion posteriorly, and the larger bronchi on both sides show some congestion, and contain some muco-pus. There is no pneumonic change, and no evidence of tuberculosis.

In neither of these two cases was there any permanent stretching of the orifice, and, to my own mind at least, it is abundantly clear that the cause of the diastolic murmur was faulty adaptation of the different cusps of the aortic valve. From the structure of the aorta, it is impossible to conceive of temporary dilatation such as may, without a doubt, occur at the pulmonary orifice, and in default of any pathological proof of enlargement of the orifice, faulty application of the individual cusps to each other can alone be entertained as the cause of regurgitation.